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Main Article

Clinical features and management of facial nerve paralysis in children: analysis of 24 cases

H E Cha, M K Baek, J H Yoon, B K Yoon, M J Kim, J H Lee

Abstract

Objectives: To evaluate the causes, treatment modalities and recovery rate of paediatric facial nerve paralysis.

Materials and methods: We analysed 24 cases of paediatric facial nerve paralysis diagnosed in the otolaryngology department of Gachon University Gil Medical Center between January 2001 and June 2006.

Results: The most common cause was idiopathic palsy (16 cases, 66.7 per cent). The most common degree of facial nerve paralysis on first presentation was House–Brackmann grade IV (15 of 24 cases). All cases were treated with steroids. One of the 24 cases was also treated surgically with facial nerve decompression. Twenty-two cases (91.6 per cent) recovered to House–Brackmann grade I or II over the six-month follow-up period.

Conclusion: Facial nerve paralysis in children can generally be successfully treated with conservative measures. However, in cases associated with trauma, radiological investigation is required for further evaluation and treatment.

Key words: Facial Paralysis; Bell's Palsy; Temporal Bone; Child

Introduction

Facial nerve paralysis is commonly due to infectious causes, trauma and middle-ear surgery. It may also be caused by tumours involving the facial nerve, congenital anomalies or systemic disease.[1,2] Facial nerve paralysis usually involves only one facial nerve, which often results in cosmetic problems as well as disordered phonation and mastication, as the condition frequently does not resolve even after aggressive treatment. This may lead to physical and psychological sequelae, with long-term, prominent deficits.

There have been many reports on the cause, diagnosis, treatment and prognosis of facial nerve paralysis in adults,[3–5] but few cases have been reported in children.

The objective of this study was to analyse the causes, clinical features, treatment modalities and recovery rate in children diagnosed with peripheral facial nerve paralysis.

Materials and methods

We retrospectively reviewed the clinical data of twenty-four children under the age of 15 years who had been diagnosed with peripheral facial nerve paralysis between January 2001 and June 2006. Children suffering from facial nerve paralysis caused by central nervous system lesions were excluded.

We undertook a retrospective medical record review to determine each child's age and sex and their facial nerve paralysis aetiology, diagnostic tools, treatment modalities and grade at initial diagnosis. Causes of facial nerve paralysis included infection, trauma, neoplasm and iatrogenic paralysis, as assessed by past history, systematic review, and serological and radiological studies. Peripheral facial nerve paralysis without an identifiable cause was classified as Bell's palsy. The grade of facial nerve paralysis and the recovery rate after treatment were evaluated according to the House–Brackmann facial nerve grading system. Using this system, a grade of I or II was defined as a satisfactory result. Treatment modalities were analysed in terms of the aetiology of paralysis. Appropriate treatment modalities were used in each case in order to maximise recovery (Table I). All patients consented to give the information of the diseases.

Results

Age and sex distribution

The 24 children were aged from three to 15 years, with a mean age of 11.3 years. There were 14 boys

From the Department of Otolaryngology – Head and Neck Surgery, Graduate School of Medicine, Gachon University of Medicine and Science, Incheon, South Korea.
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TABLE I

CLINICAL PROFILE OF CHILDREN

Pt no	Age (y)	Sex	Initial grade	Treatment	Final diagnosis	Discharge grade	Final grade
1	8	M	II	S, PT	L Bell's palsy	II	I
2	12	M	IV	S, PT	L Bell's palsy	II	I
3	16	M	IV	S, PT	L Bell's palsy	III	II
4	14	F	IV	S, PT	L Bell's palsy	III	II
5	7	F	III	S, PT	L Bell's palsy	III	II
6	7	M	IV	S, PT	L Bell's palsy	II	I
7	13	M	IV	S, PT	L Bell's palsy	IV	II
8	10	F	IV	S, PT	L Bell's palsy	IV	II
9	15	F	IV	S, PT	L Bell's palsy	IV	II
10	13	M	II	S, PT	L Bell's palsy	II	I
11	3	M	II	S, PT	R Bell's palsy	II	I
12	10	M	IV	S, PT	R Bell's palsy	IV	II
13	14	F	III	S, PT	R Bell's palsy	II	I
14	12	F	IV	S, PT	R Bell's palsy	IV	II
15	15	M	IV	S, PT	R Bell's palsy	IV	II
16	15	M	IV	S, PT	R Bell's palsy	III	II
17	15	F	II	S, AV, PT	L HZ oticus	I	I
18	14	F	IV	S, AV, PT	L HZ oticus	IV	II
19	15	M	IV	S, AV, PT	R HZ oticus	III	II
20	6	F	III	S, AB, PT	L AOM	II	I
21	9	M	III	S, AB, PT	R temp bone fx	III	I
22	3	M	IV	S, AB, PT	R temp bone fx	V	IV
23	15	F	V	S, AB, PT, OP	R temp bone fx	IV	III
24	11	M	IV	S, AB, PT	R temp bone fx	II	I

Pt no = patient number; y = year; grade = House–Brackmann grade; M = male; F = female; L = left; R = right; S = steroid; PT = physical therapy; AV = antiviral agent; AB = antibiotic; OP = operation; HZ = herpes zoster; AOM = acute otitis media; temp bone fx = temporal bone fracture

(58.3 per cent) and 10 girls (41.7 per cent). The median follow-up duration was 9.2 months (range, 6.7 to 15.2 months). All 24 cases involved unilateral facial nerve paralysis, 11 on the right side and 13 on the left.

Aetiology of facial nerve paralysis

The aetiology of the children's facial nerve paralysis included infection (four patients), trauma (four) and unknown causes (i.e. Bell's palsy; 16 patients). Peripheral facial nerve paralysis without an identifiable cause was classified as Bell's palsy. Three patients' facial nerve paralysis was due to herpes zoster oticus, and one patient's paralysis to acute suppurative otitis media. Four patients had traumatic facial nerve paralysis, all caused by temporal bone fractures (Table II). Tumours and iatrogenic causes were not encountered. No patients with bilateral facial nerve paralysis were encountered.

TABLE II

CAUSES OF FACIAL NERVE PARALYSIS IN CHILDREN

Cause	Pts	
	n	Total (*n* (%))
Idiopathic		
Bell's palsy	16	16 (66.7)
Infection		
Herpes zoster oticus	3	
Acute otitis media	1	4 (16.7)
Trauma		
Temp bone fx	4	4 (16.7)
Total	24	24 (100)

Pts = patients; temp bone fx = temporal bone fracture

All patients underwent clinical history taking, physical examination, auditory evaluation and electroneurography. Patients with otitis media or temporal bone fracture were also assessed by temporal bone computed tomography. Auricular herpes zoster suspected from physical examination was confirmed by testing for serological viral markers.

The length of time from the onset of facial nerve paralysis to treatment varied from 1 to 18 days, with a mean of 4.6 days.

Treatment

All patients with facial nerve paralysis were treated with oral steroids, ophthalmological care to protect their eyes, and facial physical therapy. The steroid dosage was similar to the Ballenger regimen, but with the dose reduced according to the child's weight. If the child weighed 20 kg, 24 mg for the first 5 days; 16 mg for the next 2 days; then 8 mg for 2 days; and family 4 mg on the last day.

Antiviral agents were also administered to three patients with herpes zoster oticus. Acyclovir (Zovi®; Korea United, Seoul, South Korea), an antiviral agent, was administered intravenously for 7 days at a dosage of 10 mg/kg/day.

Steroid therapy together with myringotomy was performed for patients with acute suppurative otitis media.

Three patients with delayed facial nerve paralysis plus temporal bone fracture were treated with oral steroids and intravenous antibiotics with secondary cephalosporin. In one patient with sudden-onset facial nerve paralysis caused by a temporal bone fracture, facial nerve decompression was performed via a transmastoid approach (Table III).

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Facial nerve paralysis grades and recovery rates

Of the 24 patients, 15 presented with House–Brackmann grade IV facial nerve paralysis (the commonest grade), four with grade III, four with grade II and one with grade V (Figure 1).

House-Brackmann grade I paralysis was observed in one patient, grade II in eight, grade III in seven, grade IV in seven and grade V in one on discharge (Figure 2).

Six months after hospital discharge, grade I House–Brackmann paralysis was observed in 10 patients, grade II in 12, grade III in one and grade IV in one (Figure 3). Therefore, 22 of the 24 patients (91.6 per cent) recovered to House–Brackmann grade II or less. Five of the 16 patients with Bell's palsy had recovered to grade II on their day of discharge, but, six months later, all 16 were House–Brackmann grade II or less. Two of the three patients with herpes zoster oticus recovered to House–Brackmann grade II or less. Of the four patients with temporal bone fractures, the three with delayed facial nerve paralysis improved to House–Brackmann grade II or less with conservative treatment, while the one patient with sudden-onset facial nerve paralysis (and grade V paralysis pre-operatively) had recovered to grade III paralysis six months post-operatively. The one patient with facial nerve paralysis caused by acute suppurative otitis media recovered completely after treatment.

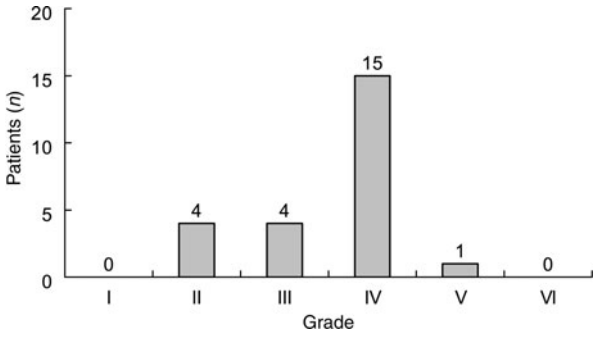

FIG. 1

Patients' House–Brackmann facial nerve paralysis grades on presentation; grade IV (62.5 per cent; 15/24) was commonest.

Discussion

The causes of peripheral facial paralysis include trauma, infection, malignancy, congenital abnormality and idiopathic palsy. According to May *et al.*,[6] the most common cause of facial nerve paralysis in children is idiopathic palsy, followed by trauma, otitis media and congenital neoplasm. Evans *et al.*[7] reported that peripheral facial nerve paralysis in children is most commonly associated with infection; other causes included trauma, iatrogenic paralysis, congenital anomaly and idiopathic palsy. In the South Korean study of Hong *et al.*[8] common paediatric causes included Bell's palsy, herpes zoster oticus, otitis media, trauma, birth injury, leukaemia and burns. In the present study, idiopathic palsy was the most common cause (66.7 per cent, 16/24), followed by temporal bone fracture (16.7 per cent, four of 24), and infections such as herpes zoster oticus and otitis media (12.5 per cent, three of 24, and 4.2 per cent, one of 24, respectively); these results are similar to other authors' findings.

In the adult population study of Park *et al.*[9] the commonest House–Brackmann facial nerve paralysis grade at diagnosis was IV (43 per cent); after treatment, this improved to grade II or less over a six-month follow-up period. Hughes[3] found that 86.4 per cent of patients with facial nerve paralysis improved to House–Brackmann grade II over a nine-month follow-up period. Hong *et al.*[8] reported House–Brackmann grade IV as the most commonly observed grade (65.3 per cent) at initial diagnosis, and found that 93.1 per cent of patients improved within six months of treatment. In the present study, 15 patients (62.5 per cent) were House–Brackmann grade IV at initial diagnosis, but 91.6 per cent had recovered to grade II or less within six months of treatment.

Danielidis *et al.*[10] reported that 78.5 per cent of their patients aged zero to nine years showed complete recovery of facial nerve function following Bell's palsy, as did 79 per cent of 10- to 19-year-olds. In the present study, all 16 children (i.e. younger than 15 years) diagnosed with Bell's palsy recovered to House–Brackmann grade II or less after steroid therapy. The one patient with acute serous otitis media recovered completely from facial nerve paralysis after treatment. Three of the four patients with temporal bone fractures, and two of the three patients with herpes zoster oticus, recovered to House–Brackmann grade II or less. Therefore, in the present study, idiopathic Bell's palsy and acute serous otitis media would appear to have better prognoses compared with other causes of facial nerve paralysis.

TABLE III

TREATMENT FOR FACIAL NERVE PARALYSIS IN CHILDREN, BY CAUSE

Cause	Pts (*n*)	Treatment
Bell's palsy	16	Steroid
Herpes zoster oticus	3	Steroid + AV
Acute otitis media	1	Myringotomy + steroid + AB + AH
Temp bone fx		
– Delayed FNP	3	Steroid + AB
– Sudden FNP	1	FN decompression + steroid + AB

Pts = patients; AV = antiviral agent; AB = antibiotic; AH = antihistamine; temp bone fx = temporal bone fracture; FNP = facial nerve paralysis

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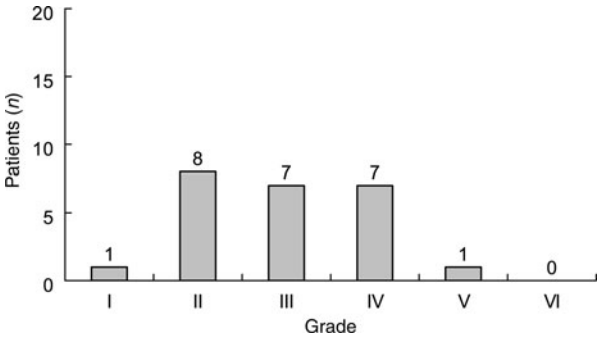

FIG. 2

Patients' House–Brackmann facial nerve paralysis grades at discharge; 37.5 per cent of patients (nine of 24) showed a satisfactory recovery at this stage.

Since the prevalence of acute serous otitis media has decreased due to antibiotic therapy and early treatment, the prevalence of facial nerve paralysis as a complication of this condition has also decreased. In the present study, only one child had acute otitis media; however, further studies with larger numbers of cases are needed to confirm this result.

Treatment methods for peripheral facial nerve paralysis can be divided into pharmaceutical and surgical intervention, but the question of which is more effective is still debated. In Engström and colleagues' study of Bell's palsy cases,[11] prednisolone shortened the time to complete facial recovery, whereas valaciclovir had no such effect. Sullivan *et al.*[12] reported no evidence of any benefit from acyclovir given alone, nor of any additional benefit of acyclovir used in combination with prednisolone. Adour *et al.*[13] proposed that, since Bell's palsy develops as a sensory neuritis due to relapse of herpes zoster virus, prednisolone therapy should be commenced within one week. However, the double blind study conducted by May *et al.*[14] found that the effectiveness of steroid therapy was statistically insignificant. Fisch[15] stated that surgical intervention should be considered in Bell's palsy patients if electroneurography detected more than 90 per cent degenerative changes within two weeks of onset. In the present study, patients with suspected Bell's palsy received oral methylprednisolone 1.2 mg/kg/day, with gradual tapering of the dose. Ophthalmological evaluation and facial physical therapy were also performed.

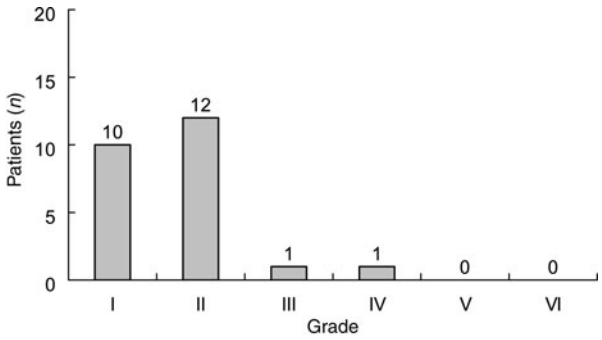

FIG. 3

Patients' House–Brackmann facial nerve paralysis grades after six months' follow up; 91.6 per cent (22/24) showed a satisfactory recovery.

Furuta *et al.*[16] suggested, in a study using polymerase chain reaction analysis, that the main mechanism of paediatric facial nerve paralysis due to herpes zoster oticus is reactivation of the varicella-zoster virus. Hato *et al.*[17] found that 78.6 per cent of paediatric patients with facial nerve paralysis recovered after treatment with steroids and antiviral agents. In the present study, patients definitively diagnosed with herpes zoster oticus (by serological viral marker study on the day of presentation) were treated with intravenous acyclovir 5 mg/kg twice a day during admission and oral acyclovir 10 mg/kg/day after discharge. As a result, 66.6 per cent of these patients recovered to House–Brackmann grade II or less patients after discharge, a slightly reduced recovery rate compared with Bell's palsy.

Mekeham *et al.*[18] found that facial nerve paralysis caused by infection (mostly herpes zoster oticus; rarely, acute or chronic otitis media) had a good prognosis and did not require surgical treatment.

Our one patient with facial nerve paralysis caused by acute otitis media (with House–Brackmann grade III at initial diagnosis) recovered completely with a combination of myringotomy and antibiotics.

Of our four patients with facial nerve paralysis caused by temporal bone fracture, three had longitudinal fractures. Of these three patients, two had House–Brackmann grade III paralysis at initial diagnosis and one had grade IV paralysis. All three of these patients showed delayed facial nerve paralysis, and had recovered to grade II or less six months after treatment.

- **This study aimed to evaluate the causes, treatment modalities and recovery rate of paediatric facial nerve paralysis, by analysing 24 cases**
- **The most common cause was idiopathic Bell's palsy**
- **Paediatric facial nerve paralysis can be successfully treated by conservative methods in the majority of cases**
- **In cases of facial nerve paralysis associated with trauma, radiological investigation is needed to enable further evaluation and treatment**

Ulug and Arif Ulubil[19] reported that 81.8 per cent of patients with traumatic facial nerve paralysis recovered to House–Brackmann grade II or I after surgery via a middle cranial fossa or transmastoid approach (the approach being based on radiological findings). They stated that the decision of whether to perform surgery or not should be based on electroneurographic findings. Yeoh *et al.*[20] found that 66 per cent of patients with traumatic facial nerve paralysis recovered to House–Brackmann grade II or I after

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facial nerve decompression via the transmastoid approach.

Our one patient with sudden-onset facial nerve paralysis caused by traumatic temporal bone fracture (who at diagnosis had House-Brackmann grade V paralysis and 95.2 per cent degeneration on electroneurography) recovered to grade III six months after facial nerve decompression plus steroid and antibiotic therapy. This suggests that surgical treatment leads to a good outcome in patients with traumatic facial nerve paralysis. In this case, facial nerve decompression via a transmastoid approach was possible because the fracture line involved the mastoid sigment of facial nerve.

In the present study, Bell's palsy was the most common cause of paediatric facial nerve paralysis, and patients improved with conservative treatment such as steroid therapy. Furthermore, we found that the recovery rate for paediatric facial nerve paralysis was similar to or slightly better than that for adult facial nerve paralysis. However, the clinical significance of these findings may be somewhat limited as this study had no control group.

Radiological studies are needed to investigate the diagnosis and treatment of facial nerve paralysis.

Facial nerve paralysis associated with trauma or otitis media can be successfully treated with a combination of steroids, antibiotics and surgery, with the decision to proceed with the latter based on electroneuroconductive studies.

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Address for correspondence:
Ju Hyoung Lee,
Department of Otolaryngology-Head and Neck Surgery,
Gil Medical Center, Gachon University,
1198, Guwol 1(il)-dong, Namdong-gu,
Incheon 405-760, Republic of Korea.

E-mail: febent@gilhospital.com

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